patients, who have a greater capacity for functional recovery than older people (who make up the bulk of our case load), in whom we doubt whether such good functional outcomes are achievable.

R J DAVENPORT
M S DENNIS
Department of Clinical Neurosciences, Western General Hospital, Crewe Road, Edinburgh EH4 2XU, Scotland, UK

Correspondence to: Dr R J Davenport
rjd@skull.dcn.ed.ac.uk

Illusory movements of the paralysed upper limb in stroke

Feinberg *et al* have recently reported the association between anosognosia for hemiplegia and the illusion of movement of the paralysed upper limb.[1] They considered the illusion a form of confabulation that is distinct from other phantom phenomena. This explanation is not supported by my findings in a patient with a stroke who experienced transient purposeful movements of his paretic hand.

The patient was a 66 year old right handed man who presented with acute onset weakness of his right arm and leg and slurring of his speech. He was known to be hypertensive and a non-insulin dependent diabetic patient. Neurological examination confirmed the presence of right hemiplegia with facial involvement and mild to moderately severe dysphasia. Muscle power, as measured by the Medical Research Council (MRC) scale, was 1/5 and 2/5 in the upper and lower limbs respectively. Spinothalamic and posterior column sensations were intact. No visual field defects were found on examination using the confrontation method. There was no astereognosis or sensory extinction of tactile or visual stimuli. The patient was alert and cooperative. His comprehension of spoken and written language was good but there was evidence of moderately severe nominal dysphasia. The rest of the physical examination was normal. Brain CT confirmed the presence of a non-haemorrhagic infarct in the left corona radiata. The patient scored 19 on the mini mental state examination. There was no evidence of hemineglect as assessed clinically and with the line bisection test. The patient was correct in 8/10 items of the anosognosia for hemiplegia questionnaire.[1]

Six weeks after his stroke the patient developed an itchy skin condition, probably a drug hypersensitivity reaction. When he scratched his skin with his left (good) hand to relieve the itching he thought that his right hand was also simultaneously scratching the same skin area. The right hand "stopped working" when he ceased scratching his skin but the perception of movement recurred each time he scratched the same or a different skin area until his symptoms resolved 2 weeks later. The use of the left hand for other activities did not result in a similar phenomenon. The patient had good insight into his motor functional disability and described his perceived hand movements as a "silly situation".

The case reported here demonstrates that illusory movements in stroke are independent of anosognosia for hemiplegia. This finding is in agreement with those of a previous study.[2] It also suggests that illusory movements are unlikely to be the product of confabulations. Confabulation is primarily a memory disorder and results from lesions in the forebrain and medial temporal lobe that disrupt connections of the limbic system.[3] The patient reported here did not have an amnestic syndrome; neither was his brain lesion (as demonstrated with CT) in the limbic system area. It seems likely that the illusory movements described by Feinberg *et al* were phantom phenomena associated with reorganisation of cortical maps and neural plasticity.[4]

A M O BAKHEIT
University of Plymouth, Mount Gould Hospital, Plymouth PL4 7QD, UK

1 Feinberg TE, Roane DM, Ali J. Illusory limb movements in anosognosia for hemiplegia. *J Neurol Neurosurg Psychiatry* 2000;**68**:511–13.
2 Lu LH, Barrett AM, Schwartz RL, *et al*. Anosognosia and confabulation during the Wada test. *Neurology* 1997;**49**:1316–22.
3 Hashimoto R, Tanaka Y, Nakano I. Amnesic confabulatory syndrome after focal basal forebrain damage. *Neurology* 2000;**54**:978–80.
4 Ramachandran VS, Hirstein W. The perception of phantom limbs. The DO Hebb lecture. *Brain* 1998;**121**:1603–30.

Feinberg and Roane reply:

The patient described by Bakheit is of interest but is not relevant to our investigation.[1] To be included in our investigation, patients were required to have *right* hemispheric strokes and *left* hemiplegia. Furthermore, 10/11 patients in our study had left hemispatial neglect and left hemisensory defect. The patient described by Bakheit had a right hemiplegia, and had *no* neglect or sensory defects. Therefore, Bakheit's patient would not have qualified for our study and cannot be fairly compared with our study population. Additionally, the factitious movements described in Bakheit's patient differed from those experienced by our patients in two significant respects. Firstly, Bakheit's patient experienced a "mirroring" phantom movement of the plegic right limb only when the normal hand was active. In our study, to minimise the potentially confounding role of completion, we specifically excluded from the main analysis those patients who only experienced illusory limb movements when the nonplegic limb was active. Secondly, the phantom movements experienced by Bakheit's patient were restricted to a particular idiosyncratic action—namely, scratching—as opposed to our patients who experienced illusory limb movements when simply asked to raise the left arm, an action which apparently failed to elicit factitious movement in Bakheit's patient. Therefore, according to the criteria set out in our investigation, the movements experienced by the patient of Bakheit would not be catagorised as illusory limb movements in our study. Finally, it should be further noted that our patients were examined within a week of onset (some within a day) of acute hemiplegia, before significant "reorganisation of cortical maps and neural plasticity" is likely to have occurred. The patient of Bakheit is reported to have had phantom movements at 6 weeks after onset of hemiplegia when cortical reorganisation and neural plastic effects are more likely to have occurred.

In our opinion, Bakheit has committed the same error that we have previously cautioned against.[2 3] He has failed to distinguish "phantom limb movements" in his patient from illusory limb movements that occur in association with right hemispheric damage and hemineglect. Patients with true phantom limbs, as in Bakheit's case, do not deny the identity of the actual arm and recognise the phantom movements as illusory. By contrast, the patients with illusory limb movements in our study all denied ownership of the plegic arm and believed in the reality of the factitious movements. It is in this group in which we found illusory limb movements and which bears a relation to anosognosia and represents a variety of confabulation. Finally, we point out that confabulation is not confined to amnestic patients, and occurs in other conditions such as Anton's syndrome.

TODD E FEINBERG
Yarmon Neurobehavior and Alzheimer's Disease Center, Beth Israel Medical Center, 317 East 17th Street, New York, New York 10003,USA

DAVID M ROANE
Department of Psychiatry, Beth Israel Medical Center, 16th Street and 1st Avenue, New York, New York 10003,USA

Correspondence to: Dr Todd E Feinberg

1 Feinberg TE, Roane DM, Ali J. Illusory limb movements in anosognosia for hemiplegia. *J Neurol Neurosurg Psychiatry* 2000;**68**:511–13.
2 Feinberg TE. Anosognosia and confabulation. In: Feinberg TE, Farah M, eds. *Behavioral neurology and neuropsychology*. New York: McGraw-Hill, 1997:369–90.
3 Feinberg TE, Roane DM. Anosognosia, completion, and confabulation: the neutral-personal dichotomy. *Neurocase* 1997;**3**:73–85.

Social deprivation and prevalence of epilepsy and associated health usage

I read the study of Morgan *et al* on social deprivation and prevalence of epilepsy and associated health usage[1] with great interest and would like to add some remarks from my experience in the most impoverished region of the United States, near the Mississippi Delta. I would caution that it is especially in a poor and traumatised population, extremely difficult to differentiate between true electrical events and non-epileptic (or pseudo) seizures.[2] We have known since Charcot about the correlation between psychological traumatic states, to which poverty is intimately related and conducive, and "hysterical" seizures.[3–5] There is a substantial comorbidity of epileptic and non-epileptic seizures.[2] In fact, what I see here in Mississippi is more often than not a mixture of both and without proper, expensive testing, such as video EEG, it is sometimes impossible to make the difference. Because of the way the data were collected, it is difficult to know from the paper of Morgan *et al*[1] whether pseudoseizures were properly taken into account when assessing the prevalence of epilepsy. The same caveat applies to the ascertainment of psychiatric comorbidity. A thorough neuropsychiatric screening of the clientele of an epilepsy clinic would disclose a much higher psychiatric comorbidity than the record linkage used here. Because of the way neurologists are trained, at least in the United States, most psychiatric comorbidity in neurology patients in general probably goes undiagnosed.

What the usage data of Morgan *et al* do show is how vain the treatment of neurological illness remains without addressing its social ecology. This certainly is true in Wales as well as in Mississippi.

M PRETER
Departments of Psychiatry and Neurology, University of Mississippi Medical Center, 713 Northwest Avenue, Durant, MS 39063–3007, USA

Correspondence to:
mpreter@psychiatry.umsmed.edu

1 Morgan CLI, Ahmed Z, Kerr MP. Social deprivation and prevalence of epilepsy and associated health usage. *J Neurol Neurosurg Psychiatry* 2000;**69**:13–17.
2 Devinsky O. Non-epileptic psychogenic seizures: quagmires of pathophysiology, diagnosis, and treatment. *Epilepsia* 1998;**39**:458–62.

3 Bowman ES, Markand ON. The contribution of life events to pseudoseizure occurrence in adults. *Bull Menninger Clin* 1999;**63**:70–88.
4 Bowman ES, Markand ON. Psychodynamics and psychiatric diagnoses of pseudoseizure subjects. *Am J Psychiatry* 1996;**153**:57–63.
5 Cartmill A, Betts T. Seizure behaviour in a patient with post-traumatic stress disorder following rape. Notes on the aetiology of pseudoseizures. *Seizure* 1992;**1**:33–6.

The authors reply:

We thank Preter for his interest in our paper and for his comments identifying the problems associated with correctly diagnosing epilepsy. As we have indicated in the paper, these problems are intensified by record linkage techniques with the possibility of both false positive and false negative results. We discussed in some detail the issue of false negatives as we think this to be the greater problem within our study and so Preter's comments about false positives, particularly pseudoseizures, are most useful. Patients with pseudoseizures, however, will still place a demand on epilepsy services and therefore remain an issue in the allocation of resources within areas of high social deprivation.

We also accept that our ascertainment of psychiatric morbidity will be skewed towards the more severe forms of psychiatric comorbidity as, by our methodology, they will have to have come into contact with secondary care services. It is, however, these patients, excluded from our second analysis, who will have the greatest influence upon social and material deprivation.

We think, however, that despite these caveats, the findings of the study remain valid. As is often the case, a record linkage study raises as many questions as it answers and more detailed research is required in this area.

C L MORGAN
Z AHMED
M P KERR
Department of Psychological Medicine, University of Wales, College of Medicine, Heath Park, Cardiff CF4 4XW, UK

Correspondence to: Dr M P Kerr.
KerrMP@cf.ac.uk

Neuropsychological abnormalities in first degree relatives of patients with familial Parkinson's disease

We enjoyed reading the paper by Dujardin *et al*[1] who investigated possible preclinical features of asymptomatic relatives in families with Parkinson's disease. A battery of neuropsychological tests disclosed impaired frontal executive function in 15 of 41 first degree relatives of patients with familial Parkinson's disease. Nine showed general frontal executive impairment. The other six only had lower scores in parts of motor dynamic sequences and word fluency. The authors concluded that this dysexecutive syndrome could be a premorbid expression of Parkinson's disease. It could represent an early nigrostriatal dysfunction in first degree relatives of probands with familial Parkinson's disease who may thus carry a higher genetic risk of developing the disease.

Dujardin *et al* describe modifications of the cognitive status which we reported in unaffected co-twins of patients with Parkinson's disease[2] After this, 3 years ago our group published a similar study[3] to the one by Dujardin *et al.* As they do not mention our findings, we briefly discuss our data in relation to their results. We compared 35 motor asymptomatic first degree relatives (mean age 52.6 (SD 10.6) years) of families with at least two members affected by Parkinson's disease to 29 relatives (mean age 52.1 (SD 4.1) years) of patients with sporadic Parkinson's disease and to 32 healthy controls (mean age 51.9 (SD 4.6) years). To account for a possible "low dopamine syndrome", we studied memory, frontal lobe function, mood, personality traits, somatic complaints, and fine motor abilities. Tests used were the short form of the Wechsler adult intelligence scale, the auditory verbal learning test, the controlled oral word association test, the Wisconsin card sorting test (Nelson version), the paranoid depression scale, the revised version of the Freiburg personality inventory, a list of complaints, and a standardised finger tapping test. We found that first degree relatives of both patients with familial Parkinson's disease and those with sporadic Parkinson's disease differed significantly from controls in several tests. They had lower scores in total fluency and fewer categories in the Wisconsin card sorting test. Relatives of both patients with familial Parkinson's disease and with sporadic disease expressed more impulsiveness, more strain, and less extraversion on personality assessment. In addition, relatives of patients with familial Parkinson's disease had more errors than controls in the Wisconsin card sorting test. Relatives of patients with sporadic Parkinson's disease showed more depression, more somatic complaints, and inhibitedness than controls and also less extraversion, less emotionality, and a lower tapping rate of the right hand. Our results, both motor and nonmotor, were comparable with those of patients with early stage Parkinson's disease and are in keeping with some of the findings of Dujardin *et al*.

On average, our proband sample was 14 years older than that of Dujardin *et al*, and by contrast with these authors, we included assessment of depression as a possible confounder of the neuropsychological test results. Depression may have a substantial impact on cognitive function,[4] and a history of depression is thought to be a risk factor for developing Parkinson's disease.[5] In our study, there were no correlations between cognitive impairment and depression. We therefore considered frontal lobe dysfunction and depression as independent signs of the "low dopamine syndrome" in our samples. Another important result of our investigation was that, apart from one personality trait ("aggressiveness"), we could not establish differences between relatives of patients with familial Parkinson's disease and those of patients with sporadic Parkinson's disease in any test item, nor were there item clusters in subsets of probands. Thus, according to our data, frontal lobe dysfunction and depression can be found to a variable degree in some relatives of patients with both the familial and the sporadic form of Parkinson's disease. It should be kept in mind that the finding of such neuropsychological abnormalities does not prove that their origin is genetic.

B KIS
I HEBERLEIN
J HAGENAH
H JACOBS
C KLEIN
P VIEREGGE
Department of Neurology, Medical University of Lübeck, Ratzeburger Allee 160, 23538 Lübeck, Germany

Correspondence to: Professor P Vieregge
vieregge_p@neuro.mu-luebeck.de

1 Dujardin K, Duhamel A, Becquet E, *et al.* Neuropsychological abnormalities in first degree relatives of patients with familial Parkinson's disease. *J Neurol Neurosurg Psychiatry* 1999;**67**:323–8.
2 Holthoff VA, Vieregge P, Kessler J, *et al.* Discordant twins with Parkinson's disease: positron emission tomography and early signs of impaired cognitive circuits. *Ann Neurol* 1994;**36**:176–82.
3 Vieregge P, Heberlein I, Kömpf D. Are neuropsychological tests useful in screening for the genetic risk of Parkinson's disease? *Parkinsonism and Related Disorders* 1997;**3**:141–50.
4 Channon S, Green PSS. Executive function in depression: the role of performance strategies in aiding depressed and non-depressed participants. *J Neurol Neurosurg Psychiatry* 1999;**66**:162–71.
5 Taylor CA, Saint-Hilaire MH, Cupples LA, *et al*. Environmental, medical, and family history risk factors for Parkinson's disease: a New England-based case control study. *Am J Med Genet* 1999;**88**:742–9.

BOOK REVIEW

Juvenile Myoclonic Epilepsy: the Janz Syndrome. Edited by B SCHMITZ and T SANDER. (Pp 207, £42.50). Petersfield: Wrightson Biomedical, 2000. ISBN 1 871816 42 4.

Have you ever had that feeling that something is just on the tip of your tongue but you can't quite get at it or that if only you had one more piece of the jigsaw, you would be able to see the whole picture? Welcome to juvenile myoclonic epilepsy. It is one of the most rewarding conditions in epilepsy to diagnose and treat. Indeed juvenile myoclonic epilepsy has the unusual, dual virtues of being both common and treatable. But what is it? This book introduces the condition—prevalence 3%-11% of all epilepsy, easily diagnosed if you think to ask for early morning twitchiness or clumsiness, characteristic EEG appearance etc. But then come all the tantalising clues that leave one on the brink of understanding. It is obviously genetic and a linkage to chromosome 6 has been suggested for years, now honed down to near the HLA gene. But a recent analysis has tried to subdivide juvenile myoclonic epilepsy according to electroclinical criteria to obtain more homogeneous groups for genetic analysis and this has suggested genetic heterogeneity. Why are there so many focal elements in this generalised epilepsy syndrome? These include focal clinical seizure manifestations, focal EEG changes, focal imaging changes such as thickening of the grey matter detectable by mathematical techniques. What is the overlap with other syndromes such as childhood absence epilepsy and why are seizures triggered by reading or praxis in some cases?

At least all can agree that it usually gets better with valproate but comes back if you stop the drug. Unfortunately this text does not discuss other newer medications. Experience with them is largely anecdotal except the treatment of the myoclonus with benzodiazepines and piracetam.

This book summarises our knowledge of juvenile myoclonic epilepsy in a readable and concise but comprehensive text. The trouble is that we are on a threshold between descriptive knowledge and understanding so juvenile myoclonic epilepsy remains one jigsaw piece short of a picture. It will be of interest primarily to those in the epilepsy and genetics fields.

MARK MANFORD